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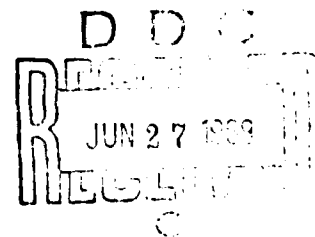
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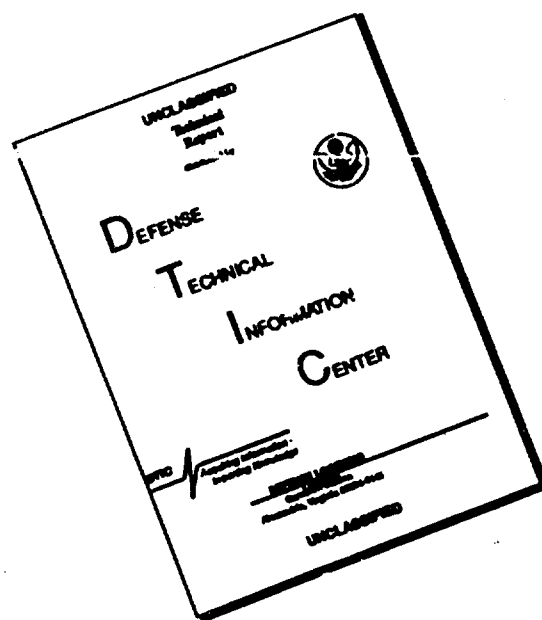
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FIRST AFRICAN CASE OF SPOROTRICHOSIS BEURMANI:
TRANSMISSION OF SPOROTRICHOSIS FROM MULE TO MAN.

Bulletin & Memoranda of the
Medical Society of the Paris
Hospitals, 28: 507-510, 1909

Author: M. CAROUGEAU,
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In Tananarive, in 1906-1907, I made a study of an epizootic affecting horses and mules. The affliction had been diagnosed both as epizootic lymphangitis, due to an infection of *Cryptococcus farcinimosus*, and as glanders.

Struck by certain clinical differences between my own observations and these two maladies, I assumed that this might possibly be another disease. I made a complete study of it, which made it possible for me to discover, in the lesions, a specific fungus, with which I was able to reproduce the disease experimentally (1). A comparison of specimens and samples soon disclosed that this was sporotrichum Beurmanii.

Equine sporotrichoses generally take one of two forms most frequently encountered in man: a disseminated gummatous hypodermic form, and a centripetal gummatous lymphangitis; occasionally, ~~you see~~ nasal and conjunctival lesions. In one case, autopsy revealed pulmonary sporotrichotic nodules.

The development of the lesions is the same as in man: a hard nodule which softens, suppurates, and eventually ulcerates. The duration of the malady is indefinite; its animal victims die of exhaustion. However, the moment we knew the real nature of the malady, we instituted the classic iodine-iodide treatment for human sporotrichosis. The effect was

electrifying. Animals covered with lesions, already slated for the slaughter, were cured in a matter of a few weeks, and ready for service.

There is no need to dwell on the economic implications of proper and swift diagnosis of sporotrichosis. With 500 grams of potassium iodide, costing 25 francs, you can save an animal whose average value is about 1,000 francs.

This particular sporotrichosis is of interest in human clinical study, since it can be transmitted from the animal to a healthy human. In fact, this first African case of human sporotrichosis which I have the honor of presenting to the Society, was a case of mule-to-man infection.

All cases of human sporotrichosis contamination from animals should be reported, since they prove the danger of such infection from domestic animals.

In their report of 1907, Lutz and Splendore comment on the case of a nurse who was bitten at night by an animal assumed to be a rat. Shortly thereafter, typical symptoms of sporotrichotic lymphngitis developed, due to *s. Beurmanii*. It is probable, although impossible to prove, since the suspected animal could not be found and examined, that this case of human sporotrichosis was of animal origin.

Lutz, as a matter of fact, discovered spontaneous sporotrichosis in rats, and the saprophytic presence of sporotrichium Beurmanii in rat mucosa.

On the subject of spontaneous sporotrichosis in dogs, Gougerot and Caraven take the diffusion patterns of sporotrichium Beurmanii as the basis for a hypothesis of animal-to-man transmission.

We have ourselves recently described spontaneous sporotrichosis in mules and horses observed in Madagascar, and due to sporotrichium Beurmanii infection.

We can report one case in which the contagion passed from mule to man. The observation is very clear, even though no culture was taken to validate it formally.

In May of 1906, M.G., a military veterinary still on duty in Tananarive, was assigned to treat some mules for sub-cutaneous eruptions then diagnosed as epizootic lymphangitis. The mules were actually suffering from sporotrichosis, as demonstrated by the fact that I first found sporotrichium in the cadavers of these very animals.

At that time, treatment was purely surgical: opening the eruptions, cauterization, disinfection of sores, etc.

While lancing one of these subcutaneous abscesses, M.G. gave himself a deep scratch on the tip of his left index finger.

This trifling wound, which he bathed in a creosote solution, bled copiously and cicatrized normally within a few days.

After twenty-five to twenty-seven days, the scar began to give pain. It developed an inflammation which increased very rapidly in severity, closely resembling a developing felon. The index finger was swollen, hot, and painful.

During the following week, an ascendant lymphangitis appeared. Symptoms included a reddish streak along the dorsal aspect of the index and the hand, running up along the forearm and the arm. One axillary ganglion swelled painfully.

The index was extremely sensitive. A needle introduced into the most swollen portion caused a flow of blood-streaked pus. The wound resulting from this slight intervention became ulcerated, then hollowed to resemble a classic inoculation chancre.

Along the the path of lymphatic inflammation, four subcutaneous gommae developed. They were not very sensitive. One was on the arm, and three on the forearm, all identical with the sporotrichotic lymphangites displayed in patients III, VI, XII and XIII as reported by de Beurmann and Gougerot.

Prolonged baths in carbolic solution, accompanied by wet compresses, attenuated the lymphangitis within two weeks. Three of the nodules disappeared entirely; the fourth opened, yielded a thick, viscous pus, then healed fairly rapidly, leaving a hardened scar, unlike the other three, which left no trace whatever.

The sore on the index finger, however, was very difficult to heal. Along its edges, little purulent pustules formed, of about the size of a pinhead, each enclosing a drop of thick pus very like what had been found in the mule eruptions. This sort of auto-inoculation from the edges of a wound was a phenomenon known in France in 1903.

Each day, seven or eight of these tiny pustules would erupt. We changed the phenol-antiseptic bandages each day, applied tincture of iodine, and so on. Burning-hot galbanum plaster

was applied directly to the wound.

Since the exact nature of the infection was not yet known, (Potassium) iodide was not given.

Cicatrization was not complete until some three months after the accident.

Since that time, there has been no relapse.

It wasn't until the end of that year (1906) that the mule malady was identified with the Paris strain of human sporotrichosis. In the gommeae preparations from M.G., researchers had seen rounded forms mistaken for Rivolta and Micellone's cryptococcus forcinosus, because they believed that the mule infection could only be a form of epizootic lymphangitis.

What they actually saw were short forms of sporotrichum.

You will recall that the same sort of error was made in America at the start. Shortened forms of sporotrichum Schenki were found in human wounds, and assigned at the time to the blastomycetae or yeast-moulds.

However, despite the lack of human cultures, this observation brooks no question, because of the presence of the shortened forms in the pus from human gommeae, and because of the positive culture results from the mule gommeae.

The observation seemed to me worthy of attention, inasmuch as it definitely establishes the vulnerability and receptivity of man to sporotrichosis of animal origin.

In this particular case, the malady remained local, and proved curable, although after a fairly long period of convalescence. It is, however, quite conceivable that, given a less robust subject, the infection might have developed into more numerous gommeae and assorted generalized phenomena. It should also be noted that the inoculation dose was very minute, which undoubtedly favored containment of the infection in the localized area.
